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PRINCIPAL INVESTIGATOR

George H. A. Clowes, Jr., M.D.

INSTITUTION

Medical College of South Carolina

SUBJECT OF THE REPORT

THE CARDIAC OUTPUT AND VASCULAR RESPONSE TO TRAUMA

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Preparing Institution: The Medical College of South Carolina

Title of Report: THE CARDIAC OUTPUT AND VASCULAR RESPONSE TO TRAUMA

Principal Investigator: George H. A. Clowes, Jr., M.D.

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Continuing the investigation of the cardiac output and vascular responses
to injury, an additional 70 patients who were operated upon or admitted with
injuries have been observed during the past year. This has been accomplished
by serial dye dilution curves and intravascular pressure measurements for
periods as long as three months. To relate these observations to the well
known metabolic alterations caused by trauma certain simultaneous blood
chemical and respiratory measurements have been made. In addition, animal
experiments have been conducted employing an implanted aortic electromagnetic
flow meter and inlying vascular catheters which permit observations in a
comfortable conscious basal condition. By this means, a comparison has been
made between the uneventful recovery from thoracotomy and that complicated
by an extensive induced abscess.

Both the clinical and laboratory studies confirm the previous obser-
vation that uneventful recovery calls forth but little more circulatory
activity than that present under basal conditions. Infection or gangrene
require a significantly higher cardiac output to avoid serious metabolic
derangements which include a progressive and eventually fatal metabolic
acidosis. Following a period of shock and low cardiac output traumatized
patients enter a period of high cardiac output associated with increased
metabolic expenditure and other aspects of the defence reaction. This has
proven particularly true following severe burns at the time sepsis appears.
Respiratory insufficiency increases this demand for cardiac output and may in
part be relieved by the use of a respirator.

In accordance with the stated objective of this project to quantitate the cardiac output and vascular response to trauma, investigation has continued both clinically and in the experimental laboratory. To relate the well established metabolic reactions (21) (25) to the pattern of cardiovascular response in the injured patient, certain biochemical studies are being made concomitantly with the hemodynamic observations. Within the period of this report (April 1, 1962 to March 31, 1963), the principal investigator and a number of his staff moved on July 1st from Western Reserve University in Cleveland, Ohio, to the Medical College of South Carolina in Charleston, South Carolina. Arrangements were made to study patients in the wards of the Medical College Hospital. Temporary chemistry and physiological laboratories were established until it was possible to move into the recently completed Research Building. This took place in November. Due to delays in the delivery of certain equipment some of the experimental and clinical observations were delayed, but others have been almost continuously pursued. Some 70 patients have been studied and 72 chronic animal experiments were carried out.

With the background of knowledge obtained in previous projects, attention has been devoted to the effects of sepsis, gangrene, and shock on the circulatory response to trauma during the period of recovery and convalescence from trauma and surgery. To increase an understanding of the metabolic relationship to the alterations in circulation induced by these conditions these studies have included observations upon the behavior of the extracellular electrolytes, blood gasses, and the catechol amine concentrations in the blood. Particular emphasis has been placed upon the differences in circulatory behavior of those patients or experimental animals who died and those which survived. From this, a pattern of recovery is becoming established for uneventful convalescence which contrasts significantly from that complicated by the presence of sepsis or gangrene. The latter stresses appear to require a much greater circulatory output over long periods than occurs when

complications are not present. When this extra circulatory demand is not
satisfied, serious progressive metabolic derangements similar to those found
in shock occur and eventually result in death.

This report serves as a supplement to those in April, 1962 and October,
1962. An attempt is not made to review the data previously collected, but
rather to point up certain new observations such as the effects of burn sepsis
on the demands for circulation. In a number of instances in which the data
collected confirm impressions concerning the circulatory responses gained in
the previous periods of this and other projects, mention is made of the older
observations. Papers are in preparation concerning certain phases of this
work. One outlining the importance of the cardiac and circulatory function
to the metabolic status following trauma is in press (10). Another is being
presented to the Federated Societies this year which deals with the physiologi-
cal experimental observations, pointing out the significant increase of
circulation required for recovery in animals with induced abscesses (1).

In the future, attempts will be made to relate the part played by the
known endocrine and metabolic behavior of the injured organism to the
continued function of the cardiovascular system. Much is known quantitatively
of these metabolic alterations, but relatively little of their influence on
the chemical or functional activity of tissues or organ systems.

METHODS AND MATERIALS

Serial observations of the circulatory status in patients included
measurements of the cardiac output, the central venous pressure, and arterial
blood pressure. A long inlying venous catheter placed through the anticubital
vein so that its tip lay in the superior vena cava or the right atrium was
employed not only to measure the venous pressure but for injecting indocyanine
green dye (13) for the measurement of cardiac output. Continuous withdrawal
of arterial blood through a densitometer for the construction of dye dilution
curves (14) was accomplished by means of an inlying cannula in the radial artery.

As many as five or six such determinations in a day were not unusual. The "Rochester needles" employed for this purpose commonly were allowed to remain in place for periods up to four days filled with a dilute solution of heparin in physiological saline. Arterial blood pressure was recorded directly through a strain gauge or by means of a sphygmomanometer.

Experimentally, in addition to the above circulatory studies, the cardiac output of dogs was recorded by means of electromagnetic flow meter transducers implanted at the base of the aorta. This permitted observations in conscious animals lying quietly on a laboratory table in a basal state. Although this electromagnetic flow meter of Olmsted's (23) design functioned well, considerable trouble was encountered with short circuits caused by leaks in the plastic coating of the wire and arterial probe. Work with the designer and the manufacturer has overcome this difficulty to a great extent. The rather high incidence of aortic necrosis and rupture (62%) previously encountered has been reduced by improvement in operating technique and suitable padding with Ivalon sponge. In the last fifty animals operated upon, this occurred but eight times. A technique for the placement of inlying catheters in the arteries and veins of the leg has made possible serial dye dilution curves as a means of checking the cardiac outputs recorded by the aortic flow meters. In addition, pressure recordings have been made directly through the vascular cannulae. All this in fully conscious, completely comfortable animals in a basal state.

Arterial blood samples from patients and animals taken at the times of the circulatory measurements have been analysed for the concentrations of hydrogen ion, sodium, potassium, chloride, lactate, pyruvate, and the blood gasses. Buffer base and other acid-base values have been measured by the method of Astrup (2). Employing the Aminco-Bowen spectrofluorimeter, a method for determination of epinephrine and norepinephrine blood concentrations (26) has been worked up and standardized in cooperation with members of the

Department of Pharmacology (Dr. James Richardson). Application of this method to urinary catechol amine excretion is being established presently. Blood cortisol determinations by a modification of the method of Farrel (12) are now being undertaken but are as yet not standardized.

Provision has been made for use of beds in the Intensive Care Unit of the hospital for metabolic balance studies in the patients under study. Water balance has been accurately followed in nearly all of the patients during the period of observation. At the present time methods for the measurement of nitrogen intake and excretion are being worked out with the assistance of the Dietary Department. To date this has not been applied to the patients being studied under this project.

Efforts at measuring oxygen consumption and respiratory gas exchange in patients under study employing a respirometer have proven difficult and of poor reproducibility. Further development of this technique employing the methods of Kinney (19) and others remains to be accomplished.

EXPERIMENTAL OBSERVATIONS

Employing the Olmsted electromagnetic flowmeter for the measurement of cardiac output, and dye dilution curves intermittently as a check, 72 dogs were observed. There were three groups: Group I, twelve which recovered uneventfully from the thoracotomy without infection; Group II, twenty in which an extensive abscess with tissue necrosis was induced in the thigh by the intramuscular injection of calcium chloride but which survived; and Group III, seventeen which died of the septic process at various times from two to twelve days. Some of the septic animals developed an empyema in addition to the induced abscess in both Groups II and III. In addition, there were 23 animals which either died of rupture of the aorta or developed respiratory complications. All of these were discarded from the experimental series.

In Figures 1 and 2 are given a comparison of the composite data on cardiac output from the animals in Groups I (non infected) and II (septic survivors). As may be seen, there is a satisfactory correlation in each group showing no significant difference between the flow meter values and those obtained by dye dilution curves.

However, as demonstrated in Figure 3, there is a marked difference between the cardiac outputs of Groups I and II. The non infected animals within two days returned their cardiac indices to the normal resting basal values. At the same time the septic animals of Group II exhibited an average cardiac index of 3.7 $L/M^2/min$. The average cardiac index of the animals with sepsis continued to rise to 4.9 $L/M^2/min$ by the seventh day, while that of the non infected animals reached a plateau of 2.4 $L/M^2/min$.

From the data given in Figure 4 it is apparent that the peripheral resistance was significantly lower in the septic group of animals. The marked elevation of the cardiac output during convalescence in the septic animals to twice the basal value cannot be accounted for entirely by the demands of the increased metabolic rate with fever. There was at most a two degree centrigrade elevation of body temperature in the septic animals. This makes it appear that, in addition to the circulatory demands of extra respiratory work, there was a marked decrease in vascular resistance, probably in the vicinity of the hot inflammatory process.

It is noteworthy that neither in Groups I or II were there significant deviations from normal metabolic values. A portion of these data is given in Figure 5. The same was true of pyruvate, lactate, and the other values measured in the blood. On the other hand, septic animals which failed to maintain a high output, even though it was equal to the normal basal values of non infected animals, went into a state of shock. They died, as illustrated by the example from Group III in Figure 6, with a metabolic pattern resembling that of animals in hypovolemic shock (24). Typically there was a progressive

uncompensated metabolic acidosis.

An interesting observation was made a number of times. When an animal ruptured and drained an abscess, the circulatory alterations occasioned by the septic process returned toward normal. This is clearly demonstrable in the course of the animal given in Figure 7. For comparison the course of another animal from Group II with undrained sepsis is shown in the same chart.

CLINICAL OBSERVATIONS

Since April 1, 1962, both in Cleveland and since removal to Charleston, some 325 individual observations have been made upon 70 patients. The patients studied have included:

Trauma	8
Burns	7
Hemorrhage	3
Postabortion	3
Peritonitis	4
Operative and Postoperative	39
Miscellaneous	5

This has permitted study of the following clinical entities of interest in this project, at times two or more presenting in the same patient:

Shock:	Hypovolemic	7
	Septic	4
	Burn	7
Sepsis:	Postoperative	5
	Post traumatic	3
	Burn	7
	Peritonitis	3
	Pancreatitis	1
Major trauma and fractures		12
Respiratory failure		6
Cardiac failure		7
Uneventful convalescence from major surgery		24

At the time of entry patients with acute situations such as major burns or trauma selected for study had arterial and venous catheters placed. As soon as practicable, while treatment was being instituted, a preliminary series of hemodynamic observations was obtained including determinations of the cardiac output by dye curves. At the same time, blood samples were obtained, centrifuged, and frozen for subsequent analysis. Acid-base measurements were carried out at once, and were often an aid in therapy. Subsequently, all observations were repeated as indicated. Base line values were obtained when possible following recovery, as long as four months later in certain of the burn patients.

Patients who were to undergo elective operations and who were selected because of the magnitude of the operation or for reasons of presenting debilities had basal observations made prior to the procedure. Subsequently, these were repeated at intervals during the postoperative period.

RESULTS

The observations upon patients who underwent major abdominal surgical or thoracic operations excluding cardiac procedures, with uneventful recovery have been continued. Their data are presented in Table I. The twelve added this year serve further to confirm the pattern previously established. It appears that convalescence without the complications caused by sepsis, gangrene, or respiratory insufficiency requires but a very slight increase (18%) of circulation above the basal values to maintain a normal metabolic status in cellular respiration and energy production as reflected in the extracellular fluid. It is noteworthy that after the operations in which some degree of metabolic acidosis always developed, there was none of any significance in this group. Neither the blood lactate nor pyruvate concentrations deviated in the postoperative phase from the normal values.

By contrast, there are now studies of ten patients who developed sepsis and inflammation, but who went on to recovery. These included empyema, wound

infection, peritonitis, retroperitoneal abscess, and ischemic gangrene of the
intestine. Reference to Table I shows that on the average these patients
maintained cardiac outputs 60% above the normal basal values. There is a wide
range of variation in this response. The combined data fail to convey the
marked rise which is usually seen in the individual patient as sepsis develops.
Despite fevers which ranged from 38.9 to 41.0°C, these people did not develop
metabolic disturbances of any consequence. The oxygen consumption of this
group rose to 148% of the expected basal value, but as previously mentioned
these data are at present considered unreliable. However, as measured by
minute volume exchange, the respiratory work increased to 165% of the basal
value.

Nine patients died with sepsis or gangrene while under observation. An
example is given in Figure 10. In seven a normal period of convalescence
started. With the onset of sepsis there was a moderate elevation of cardiac
output. In three this proved inadequate to prevent the development of moderate
metabolic acidosis (buffer base deficit up to -6 mEq/L). Four others failed
to raise the outputs above their resting values. Two of these were later
proven to have old myocardial fibrosis secondary to coronary atherosclerosis.
Two patients with severe sepsis died suddenly with heart failure having
maintained outputs of 3.8 and 4.6 L/M^2/min two days prior to death. Autopsy
disclosed an acute coronary infarction in one. As shown in the table, each
of the patients in whom an observation was made within 24 hours of death had
developed a metabolic acidosis, in some instances uncompensated by respiratory
alkalosis.

Because of the variations in response it is as yet impractical to present
the data from all of the patients in the various groups. Typical examples
from each category of the situations observed are given with certain general
conclusions drawn from each group as a whole.

BURNS

Seven patients with severe second and third degree burns (estimated 34% or more of body surface) were studied this past year. In addition, two others were observed last year. From the data obtained in this group a typical pattern of cardiovascular response to this form of trauma is emerging which will form the substance of a report to be published when the number of observations becomes sufficient to be significant. An example is given in Figure 8.

The initial studies were made in four of these patients early enough that fluid shifts and circulatory changes were minimal. The cardiac indices at that time ranged from 2.8 to 5.9 $L/M^2/min$. Subsequently, the cardiac output in all but one fell to values below the normal resting value despite fluid therapy. This was accompanied by an increase of peripheral vascular resistance. As the shock phase developed an elevation of hematocrit and very low outputs ranging from 0.4 to 3.6 $L/M^2/min$ were observed. Under these conditions, the buffer base deficit increased to values ranging from -7 to -25 mEq/L, accompanied by declines of the arterial blood pH to as low as 7.07. The majority did not fall below 7.18. In all but one instance, the arterial pCO_2 was reduced to 35 mm Hg or below. One patient whose pCO_2 rose to 58 mm Hg and who had suffered a respiratory burn was placed upon a respirator with an immediate improvement in the metabolic and circulatory states. As the pCO_2 returned to 40 mm Hg and the oxygen saturation to 85%, the pH rose to 7.32 and the cardiac index to 4.1 $L/M^2/min$.

One patient with severe burns died within 24 hours. His cardiac index fell progressively to 0.4 $L/M^2/min$ shortly before death with a high central venous pressure. The metabolic acidosis increased to a buffer base deficit of -25 mEq/L and the arterial blood pH fell to 6.93 despite a pCO_2 of 32 mm Hg. The remainder of the burned patients survived five days or more. Within 24 hours the cardiac index of each was within the normal basal range, 2.4 to 4

$L/M^2/min$. One died at five days, another in seven, and one at ten. All of the patients began to exhibit cardiac outputs ranging from 30 to 115% above their expected or measured basal values after five days. This was concurrent with the onset of surface infection and hyperpyrexia. Thereafter, until the wounds were covered and the infection was brought under control the cardiac outputs remained elevated. However, none of the patients, under these conditions, showed evidence of metabolic derangements as evidenced by elevated lactates, pyruvates, or buffer base deficits. Shortly before death the cardiac index of the patient who died at five days was found to have fallen to 2.1 $L/M^2/min$ at a time when the remainder exhibited outputs ranging from 3.5 to 4.9 $L/M^2/min$. His pCO_2 climbed to 80 mm Hg and his arterial blood pH fell to 7.06. A respirator improved the acidosis but the cardiac output remained low with progressive metabolic derangements.

Another patient, who had been doing well with an output of 5.6 $L/M^2/min$ on the seventh day, suddenly dropped his output and died in heart failure with an output of 2.8 $L/M^2/min$. At the same time, he exhibited a rapidly progressive acidosis.

The patients who recovered and finally were grafted and discharged all exhibited a gradual return toward normal of their cardiac outputs. In one of these people a cardiac index amounting to more than 4.5 $L/M^2/min$ was maintained for more than six weeks before return to the normal basal value near 3 $L/M^2/min$.

TRAUMA AND HYPOVOLEMIC SHOCK

During the past two years, twelve patients in hypovolemic shock have been observed. An example of the data obtained is given in Figure 9. This patient, having bled massively during and after an extrapleural pneumonectomy, required some 18 pints of transfused blood, more than twice his normal blood volume. Three hours following his operation he exhibited a hypotension of 70/50 mm Hg with a decline of his cardiac index to 1.9 $L/M^2/min$ At that time his blood lactate had risen to 42 mgm %, and the blood pyruvate to 7 mgm %.

This was accompanied by a moderate metabolic acidosis and a buffer base deficit of -5 mEq/L. Subsequently, with arrest of hemorrhage and correction of blood volume he went on to a normal postoperative response with an elevation of the cardiac index to 3.6 and subsequently 4.2 $L/M^2/min$. With this cardiac output he maintained a normal metabolic status and went on eventually to satisfactory recovery.

Another far more complicated picture is given in Figure 10. This is the course of a 39 year old man who suffered severe multiple injuries of the chest wall, spleen, the vena cava, and a fracture of the left femur. On entry, he exhibited a combined respiratory and metabolic acidosis, accompanying both insufficient respiration and a cardiac output below 1 $L/M^2/min$. At that time, his blood lactate was over 60 mgm % while the pyruvate was but 6 mgm %. As his respiratory status was restored with the help of a respirator, and as his cardiac output returned toward normal with multiple transfusions, the metabolic acidosis and lactate values were greatly reduced. Because of oliguria on the third day and twice more thereafter he underwent hemodialysis. Apparently having started to make a recovery despite a continued slightly low cardiac output, he developed an abdominal abscess. His peripheral vascular resistance began to decline with a moderate rise of cardiac output. This was inadequate to maintain a normal blood pressure. After drainage of the abscess on the thirty-first day, he failed to meet the challenge of extra requirements for cardiac output. He died shortly thereafter with a rising venous pressure, in heart failure and a recurrence of the previous lactacidemia.

In Table I are presented the data from eight patients who underwent severe mechanical trauma or gunshot wounds but recovered. It is noteworthy that despite the absence of serious sepsis in any of these patients a large cardiac output was present as soon as the patients recovered from the shock state associated with the hypovolemia attendent upon most of these injuries.

On the second day after injury the cardiac indices ranged from 2.9 to 5.3 L/M^2/min. The patient with the greatest output had a concomitant respiratory insufficiency caused by multiple rib fractures. When placed upon a respirator and relieved of the extra work of breathing, his cardiac index was reduced to 4.1 L/M^2/min. At the same time, his buffer base deficit declined from -12 to -7 mEq/L as the arterial blood oxygen saturation rose from 68 to 91%.

Subsequently, during the first week the traumatized patients maintained elevated cardiac output values ranging from 3.9 to 5.6 L/M^2/min. In general, it might be said that the response, as indicated by the fever, leucocytosis, respiratory activity, as well as the cardiac output, was related to the extent of the soft tissue injury.

Although the data concerning epinephrine and norepinephrine concentrations in the blood are somewhat fragmentary until recently, it has become apparent that total values as high as 12 micrograms are present at times, particularly when acute vasoconstriction with high peripheral vascular resistance are present.

DISCUSSION

The data obtained confirm the impressions previously gained (6) (7) (8) (9) (10) concerning the normal cardiovascular responses during convalescence from major surgical operations. It appears that in the absence of complications little more than the basal resting circulatory output is needed to maintain a normal metabolic status. On the other hand, both the experimental and clinical observations herein reported indicate that in the presence of severe sepsis and inflammation a considerably greater cardiac output is required to maintain the normal cellular respiratory activity without the development of metabolic acidosis, lactacidemia, and other evidence of anaerobic glycolysis.

The first major experimental program of this project is now nearly complete with satisfactorily reproducible results. These are given in Figures 1, 2, and 3, indicating that the dog exhibits a circulatory pattern during recovery from thoracotomy which resembles that of man. Furthermore, in this animal, the presence of severe sepsis produces a significant increase of cardiac output until such time as the septic process is resolved. This technique will make it possible to examine individually the parts played by the nervous (18), endocrine (15), and respiratory (20) systems in evoking this reproducible response. Thus, the door is opened to improvements in the therapy of trauma and sepsis by a greater understanding of the demands made upon the organism.

It is proposed as time goes on to conduct similar experiments in sympathectomized animals and in those subjected to spinal cord section. By denervating the limb in which an abscess is established it is expected that the influence of the afferent nerve impulses can be evaluated in terms of establishing the alarm reaction. Adrenalectomized animals may be employed to determine what part the corticoid secretion may play in permitting these cardiovascular reactions to take place in the face of adverse circumstances of acidosis and oxygen lack (5) (9) (22).

The question as to why the cardiac output is elevated with a decrease of the peripheral vascular resistance under these conditions may be partially answered in terms of increased cellular demands for oxygen and metabolic substrates. Yet, according to the formulae of Dubois (11) concerning the changes in oxygen consumption in fever, the data presented by Kinney (19) on oxygen consumption, and these data on cardiac output suggest that the oxygen and circulatory demands exceed that which would be predicted by the fevers of both patients and dogs. Two other possibilities exist. The first is that the increased work of respiration requires excess output to satisfy the demands of the muscles of respiration. This can be accounted for by the

decrease in cardiac output seen when a patient is relieved of respiratory work
by a respirator. The second possibility, related to the circulatory system
itself, is that the vascular resistance is greatly reduced in the region of
an inflammatory reaction (25). The heat of an inflammatory area may indicate
that much of the blood passing through it may actually represent an A-V shunt.
This, of course, lowers the peripheral vascular resistance in a fashion which
requires either that the resistance be increased in other areas or that the
cardiac output be increased if blood pressure is to be maintained. Since
blood pressure, in all of these studies, both in the dog and in man, appears
to be very closely guarded as long as compensation is possible, a metabolic
price must be paid in terms of tissue perfusion under conditions which
prohibit an increase of cardiac output. This phenomenon is clearly seen in
certain of the patients who in the presence of sepsis or trauma were unable
to raise their cardiac outputs above the resting values. Metabolically they
resembled the patients who were in shock or those with the "low output syndrome"
following cardiac surgery (3) (9).

The mechanism by which cardiac output is increased without an addition
to the blood volume has been postulated as being related to tissue demands (17).
The accumulation of metabolites probably leads to a decrease in the vascular
resistance of the affected areas, overcoming locally the tonic arteriolar
vasoconstrictor activity of the sympathetic nervous system. The slight
resulting variations in blood pressure set in action reflex arcs initiated
in the various baroreceptors of the aorta, carotids, and other great vessels
(16). These in turn are believed to be responsible for increased venous
tone and venous return, augmented heart action, and a greater cardiac output(4).

In man, any part of this mechanism may fail, but commonly the heart
appears to be the least able to continue in the face of a prolonged demand for
a cardiac output well above the normal resting value. This is particularly
true in older people. A number of the patients, who were apparently making

a good response to severe sepsis or tissue trauma, died rather suddenly when cardiac output declined as a result of coronary insufficiency or heart failure from other causes. Others developed a progressive metabolic acidosis while maintaining a cardiac output quite adequate for uncomplicated recovery. It was equally true that dogs who failed to maintain an elevated cardiac output in the presence of an induced abscess or empyema died in a state of progressive metabolic acidosis.

Moderate degrees of respiratory insufficiency are accompanied by increased activity of the cardiovascular system. The reverse is also true. Thus, it has become apparent that the use of the respirator with an improvement in blood oxygen saturation and restoration of the pCO_2 to the normal range not only reduces the tissue demands for circulation, but also can relieve the circulation of the demands made by the muscles of respiration in responding to increased respiratory work.

Little has been said in this report concerning the effects of bacterial endotoxin and other toxic effects on the circulation resulting from infection or gangrene within the body. Four patients in so called septic shock have been observed, but the results were equivocal, two of the patients apparently dying of heart failure and two apparently from failure of venous return. Further data will be required from observations of this phenomenon to assess this aspect of trauma on the circulation.

The effects of a variety of influences on the cardiovascular response to trauma have been studied, and a pattern of this response in uncomplicated recovery and that associated with sepsis is emerging. However, much remains to be accomplished in relating the behavior of the circulation as a whole to the known metabolic, endocrine, respiratory, and renal responses of injury.
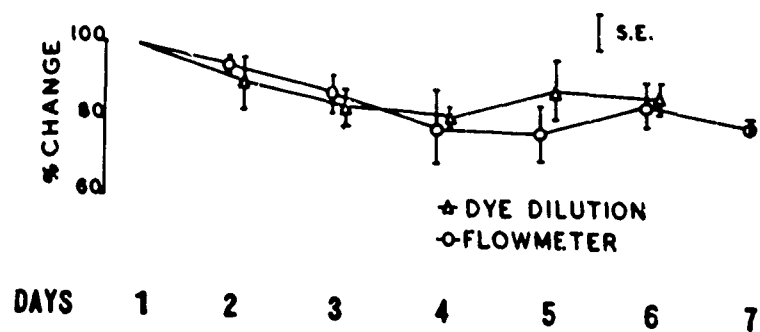
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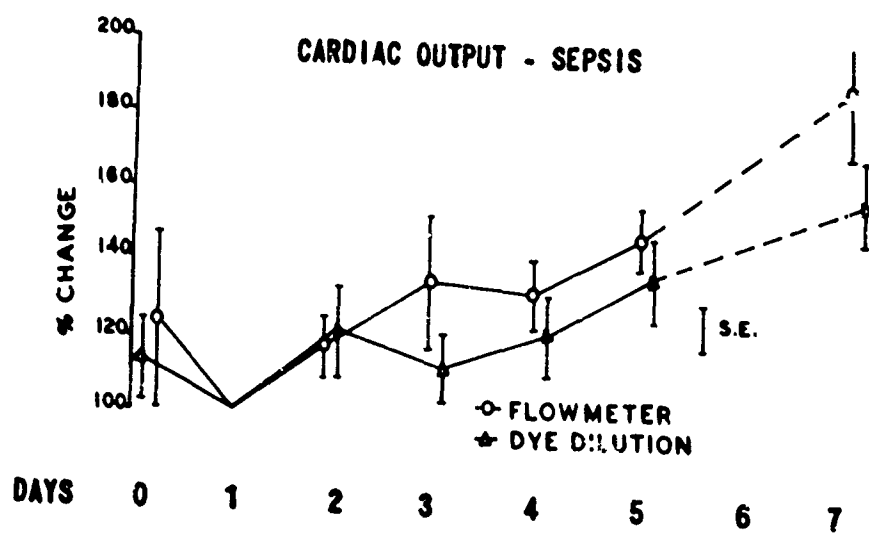
HEMODYNAMIC AND METABOLIC ALTERATIONS IN PATIENTS

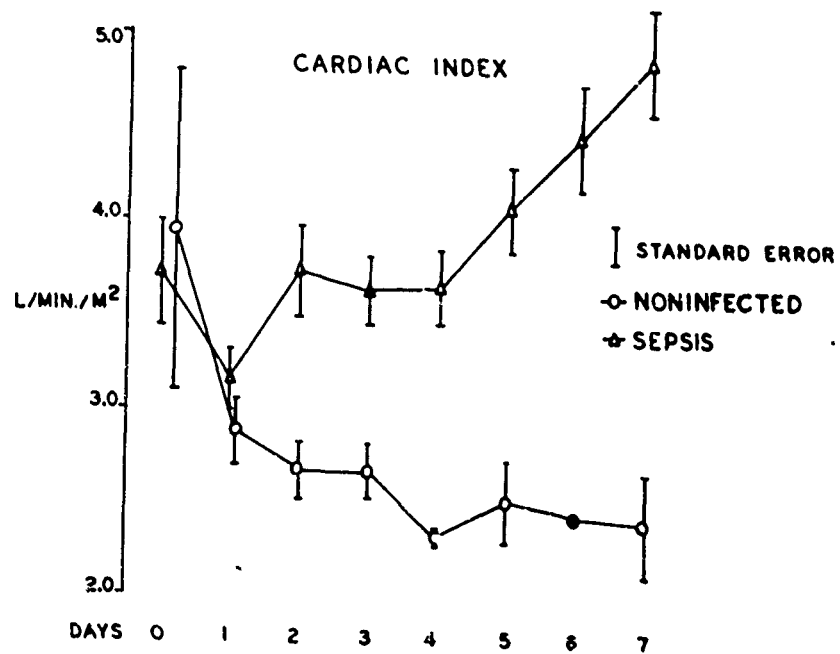| Number of Patients	Period of Observation	Cardiac Index L/M²/min Average and S.D.	Central Venous Pressure cm H2O	Arterial Pressure mm Hg	Arterial Blood pH	Buffer Base Deficit mEq/L	Lactate mg%	Pyruvate mgm %	Blood** Catechol Amine Micrograms
Normal Recovery Major Surgery 42*	Preoperative	2.7±0.4	5.1	124/75	7.41	-1	12	2.9	0
	Postop. 1 day	3.0±0.5	4.0	128/70	7.39	-3	15	3.4	1
	1 week	3.2±0.7	4.8	122/70	7.42	+2	11	3.3	0
	2 weeks	3.0±0.3	4.9	122/71	7.41	+1	10	3.0	0
Sepsis and Inflammation with Recovery 10*	Preoperative or initial	2.3±1.2	4.0	115/65	7.37	-3	10	3.1	0
	Postop. 1 day	3.1±0.6	5.2	123/72	7.38	-2	18	3.9	2
	1 week	4.3±1.4	5.5	119/65	7.41	-3	22	5.1	2
	2 weeks	3.5±1.3	4.3	115/68	7.42	+1	17	3.6	0
Sepsis and Inflammation with Death 9*	Preoperative or initial	2.6±1.4	5.9	127/69	7.38	-2	13	3.4	0
	Postop. 1 day	3.3±1.0	6.1	125/72	7.39	-4	19	4.8	0
	Prior to death 1 day	3.0±2.1	7.3	122/70	7.36	-4	25	5.1	3
	Prior to death	2.1±0.7	11.9	102/68	7.34	-8	39	6.2	8
Severe Trauma with Recovery 8*	Initial	1.4±0.8	2.2	85/58	7.31	-9	32	8.1	8
	Postop. 1 day	2.8±0.7	5.8	106/68	7.38	-5	24	6.3	0
	Postop. 1 wk	4.5±1.3	5.1	112/68	7.40	+2	20	4.2	2
	2 weeks	3.4±1.0	5.6	115/71	7.42	+1	18	4.0	0

* Data combined with those of previous report period

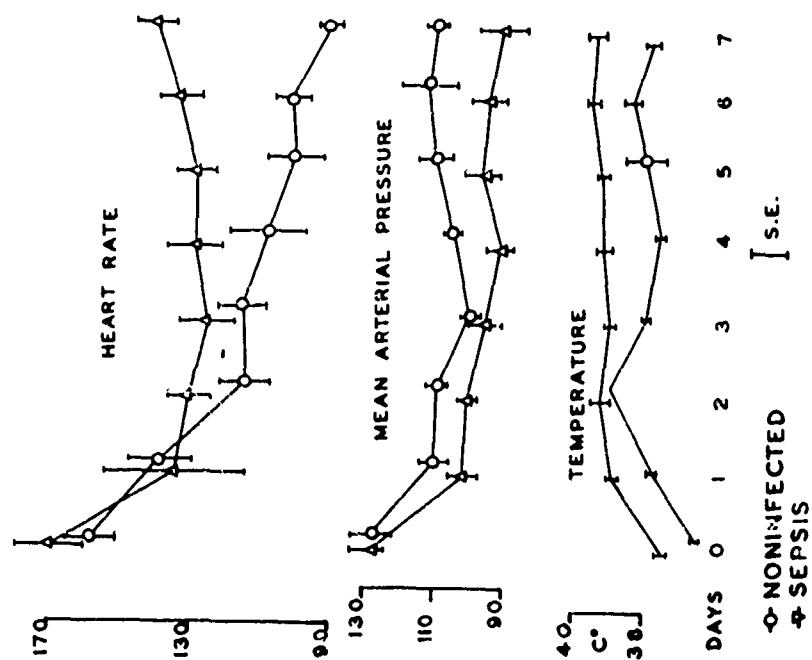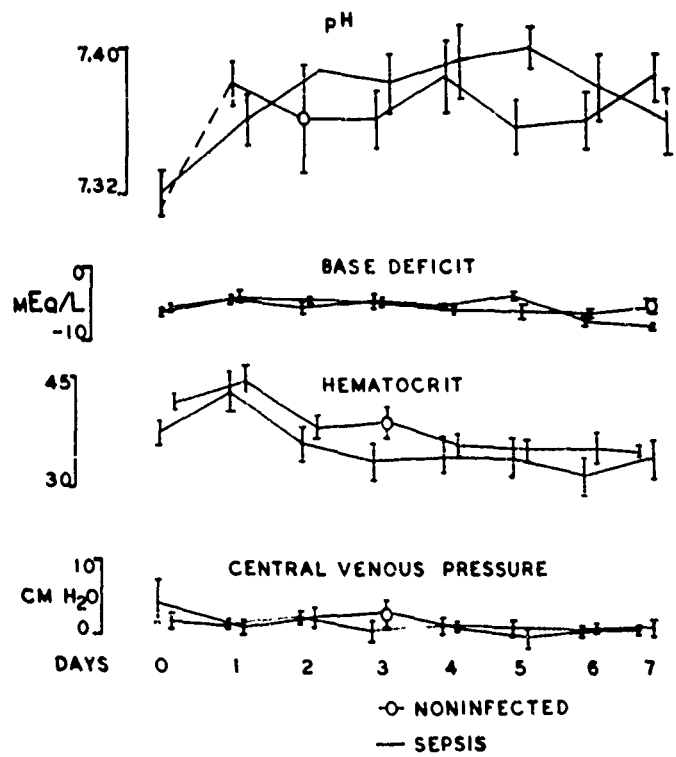** Limited number of observations makes these data insignificant
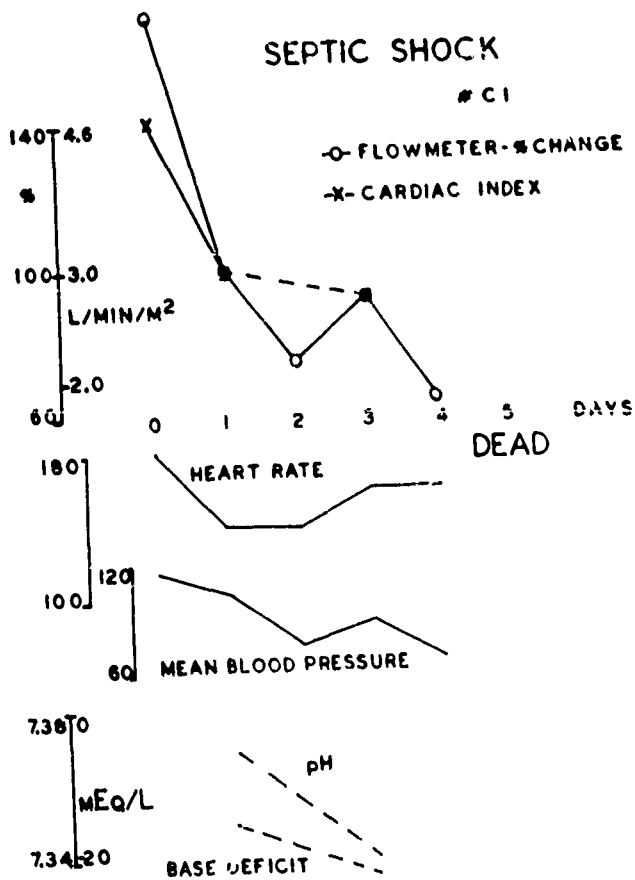
CARDIAC OUTPUT - NONINFECTED DOGS

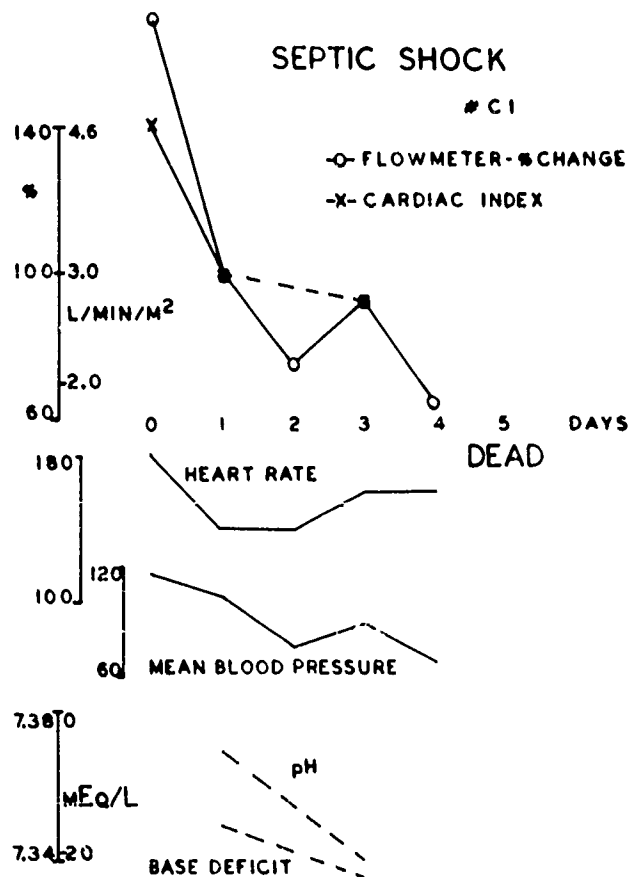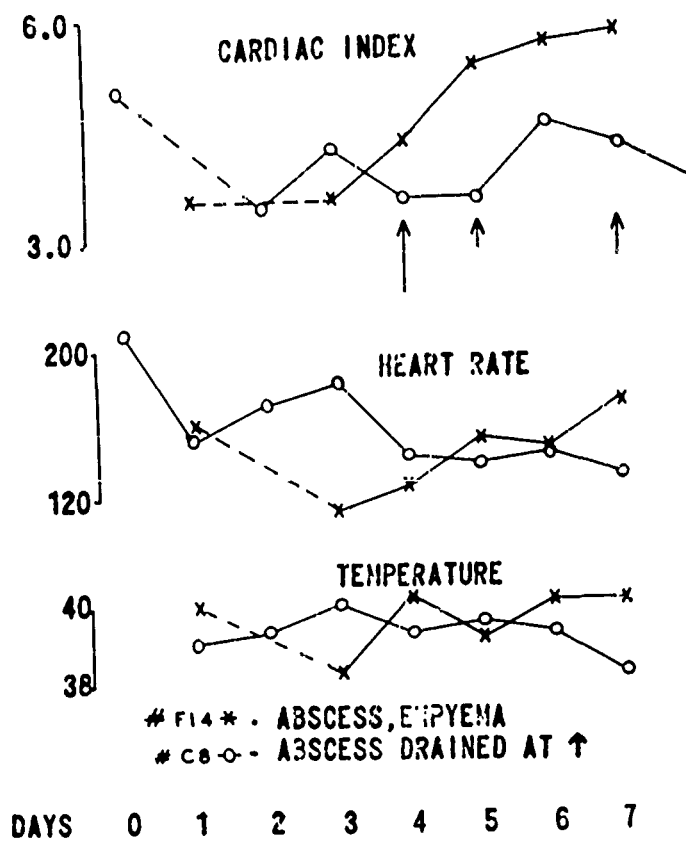CARDIAC OUTPUT - SEPSIS

CARDIAC INDEX

L/MIN./M²

STANDARD ERROR
NONINFECTED
SEPSIS

DAYS 0 1 2 3 4 5 6 7

HEART RATE

MEAN ARTERIAL PRESSURE

TEMPERATURE

170
130
90

130
110
90

40
C°
38

DAYS 0 1 2 3 4 5 6 7

◇ NONINFECTED
✦ SEPSIS

[S.E.

pH

7.40
7.32

BASE DEFICIT

MEq/L
σ
-10

HEMATOCRIT

45
30

CENTRAL VENOUS PRESSURE

CM H₂O
10
0

DAYS   0   1   2   3   4   5   6   7

-O- NONINFECTED
— SEPSIS

SEPTIC SHOCK

C 1

-O- FLOWMETER-%CHANGE

-X- CARDIAC INDEX

HEART RATE

DEAD

MEAN BLOOD PRESSURE

pH

BASE DEFICIT

SEPTIC SHOCK

C I

-O- FLOWMETER-%CHANGE

-X- CARDIAC INDEX

DEAD

HEART RATE

MEAN BLOOD PRESSURE

pH

BASE DEFICIT

CARDIAC INDEX

HEART RATE

TEMPERATURE

F14 * · ABSCESS, EMPYEMA
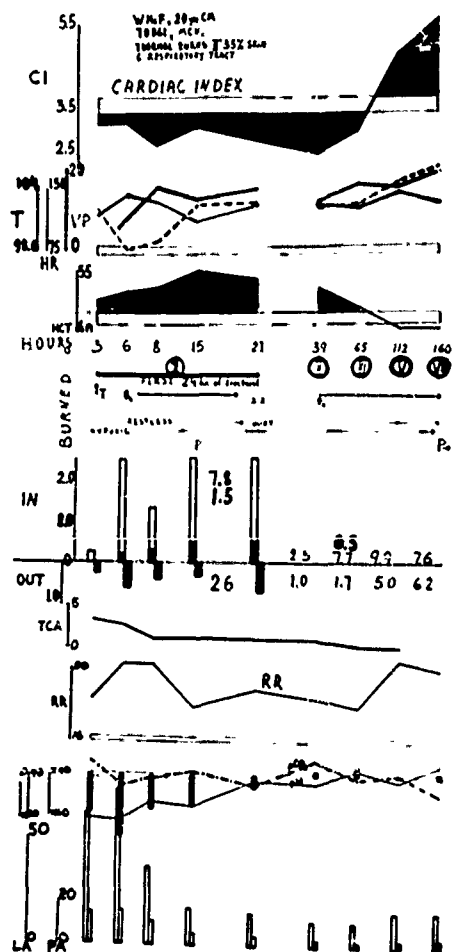# C8 -o- · ABSCESS DRAINED AT ↑

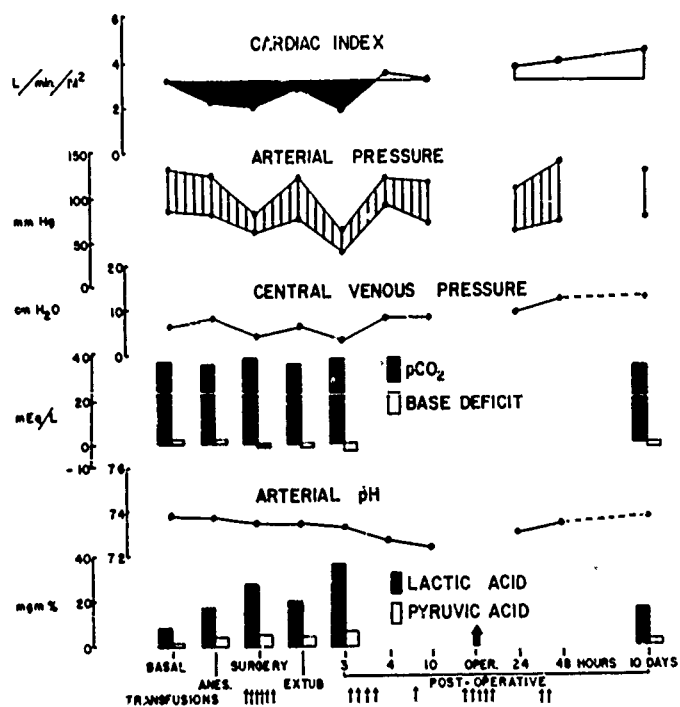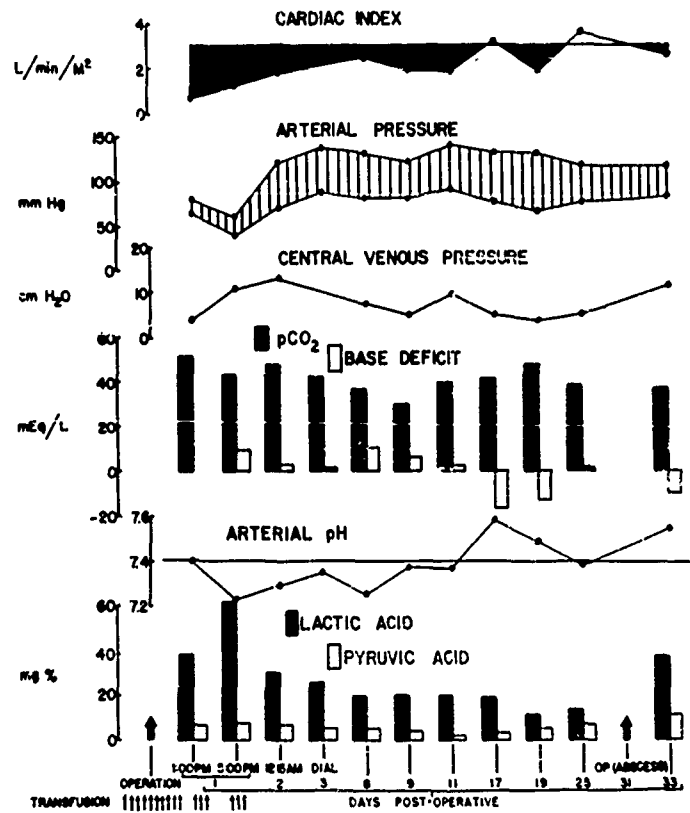DAYS   0   1   2   3   4   5   6   7

OLIGEMIC SHOCK

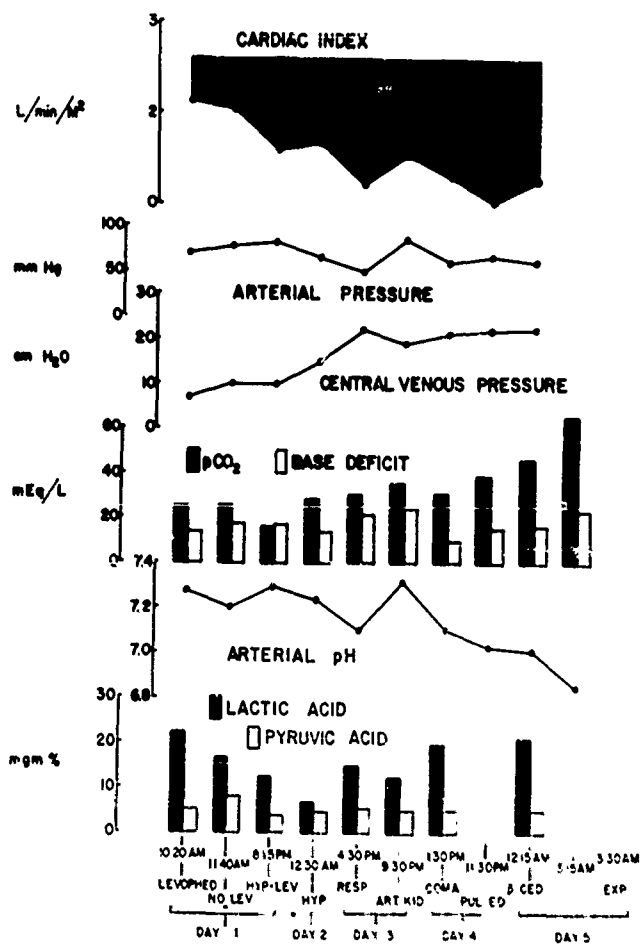EXTRAPLEURAL PNEUMONECTOMY   51YR M.   J.B.G. CMGH 10-53-52

TRAUMATIC SHOCK

MULTIPLE INJURIES   39YR M   J.S. MCH 69806

SEPTIC SHOCK

SEPTICEMIA & ABORTION   27 YR.F   S.B. MGH 70078

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